

and numbers of times that each chigger walked and/or crossed over the repellent treated area in a given time were the criteria used to evaluate repellent efficacy. The third experiment is known as the “toothpick/Q-tip (Cotton-bud) experiment”. Either toothpick and/or Q-tip (cotton bud) were soaked up with tested-repellent dilutions and later were placed in the middle of charcoal-substrate in the chigger-rearing plastic vial. The repellent zone was also designated on the side plastic vial. An un-infected *Leptotrombidium* chigger was released into the vials. Reactions and behaviors of chiggers approaching the tip of toothpick or the cotton bud as well as the repellent zone on the vial were observed and recorded. Our results revealed that the ranked efficacy of the different repellents using this system was: DEET = AI3-28724-A > AI3-26929 > DEPA.

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PATHOPHYSIOLOGICAL CHANGES IN A MURINE MODEL OF BETA-THALASSEMIA/HEMOGLOBIN E

Wannasuphaphol B, Khongnium W, Chumworathayee W, Warnnissorn M, Kalpravidh R, Pattanapunyasat K, Jamsai D, Ioannou PA, Van Gessel YA, Winichagoon P and Fucharoen S

A novel C57BL/6 transgenic murine model of the Hb E thalassemia ($\beta^{m+}/\beta^{m+}; \beta^{hE}$), which showed a normal phenotype, was previously generated. To develop the anemic model mimicking the patients, Hb E transgenic mice were bred with β -knockout mice (β^{m+}/β^{m0}) to produce double heterozygous ($\beta^{m+}/\beta^{m0}; \beta^{hE}$) and β -thal/Hb E rescued mice ($\beta^{m0}/\beta^{m0}; \beta^{hE}$). Previous hematologic study showed that rescued mice developed variable degrees of anemia similar to β -thalassemia in human. In this study, we further define hematologic properties and clinicopathologic changes in these mice. Although double heterozygote was on the β -knockout background, its phenotype was definitely normal as resulted from Hb E transgene function. Rescued mice expressed variable thalassemic phenotype due to they copy numbers of β^{hE} transgene. The slightly increased oxidative stress of the red blood cells (RBCs) was observed as well as the percentage of RBC microvesicles. RBC survival study demonstrated that mean half-time ($T_{1/2}$) and life span of rescued RBCs were definitely decreased to the same level as β -knockout mice. At necropsy, splenomegaly and hepatomegaly were present in rescued mice but not in double heterozygotes. Histologic examination of spleen and liver of rescued mice revealed iron accumulation and variable degrees of increased extramedullary hemopoiesis. These results indicated that β -thal/Hb E rescued mice that have β^E -transgene under homozygous β -knockout background developed pathophysiologic changes similar to human β -thalassemia disease. Study of this murine model will further elucidate the pathogenesis of β -thalassemia and enable us to test new therapeutic regimes, such as γ -globin-stimulating agents, antioxidants, iron chelators and gene therapy. This study was supported in part by Thailand Government Research Fund 2004 to P.W. and Thailand Research Fund to S.F. as a Senior Research Scholar.

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